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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Progress in Hospital Treatment

OSKAR DIETHELM, M.D.
New York, New York

Evaluating progress in treatment forces us to assume a humble, critical attitude. It is easy to recognize the great advances made over a long period of time as well as the mistakes which delayed or sidetracked progress but it is difficult to apply the same perspective to the present. The great value of an historical understanding is that it enables us to learn from the experiences of many others and enables us to avoid many mistakes.

Medical treatment, and above all, psychiatric treatment is dependent on scientific progress as well as on current attitudes in general. When this hospital was conceived and when it was opened, American psychiatry was in its beginning, but it was a state of great activity, with an urgent desire of the medical profession to build hospitals which would compare with those in England and on the European continent. Certain architectural principles which seemed to take care of the special needs of psychiatric patients were established. It was recognized that the buildings should not merely serve for protection of the suicidal patient and of society but that they should be attractively furnished and interestingly landscaped. The location should be desirable esthetically and in a central position of the population it would serve. It is of importance to follow these special aspects as they have evolved in the second half of the century which we are considering. A strong tendency developed in this country to stress security above all other considerations. Many modern hospitals are therefore depressing or revolting to patients who feel that the whole hospital arrangement stresses that they are rather inmates of an institution than patients in a hospital. The location has often been guided by the availability and not the suitability of land, and it had been forgotten that the patients look out of their windows and react to the attractiveness or unattractiveness of the world outside.

In the period of the opening of this hospital, wide halls and high ceilings were considered attractive and desirable. Our taste has changed and modern hospitals try to use space more efficiently. Heating and plumbing facilities have improved greatly, and the entire housekeeping routine leads to a more hygienic and more agreeable life in the hospital. Not as great strides have been made in the interior decorating which a hundred years ago was considered important by some psychiatrists who, however, had difficulty in finding a suitable solution. It was believed necessary to have indestructible furniture which at the same time would not serve as a dangerous weapon in the hands of an excited patient.

The solution to this problem did not come from improvements in building construction but from the understanding gained in the development of dynamic psychopathology and clinical psychiatry. Although since the beginning of the century, each patient was recognized as an individual whose personality

Address delivered at the Centennial Celebration, Central State Hospital, Nashville, Tennessee, Nov. 1952.

greatly affected his reaction during his illness and the pattern of his psychopathologic disorders, one still felt that his reactions, especially if he suffered from a schizophrenic illness, were not predictable and relatively little influenced by his environment. There were two valuable psychopathologic concepts which, being misapplied, impeded treatment —the concepts of autism and regression. Autism, for which other concepts were substituted, meant a shutting off of the outer world, an invisible glass wall between patient and other people, a psychotherapeutic inaccessibility. This concept led to a therapeutic hopelessness and stultified the physician's and nurse's imagination and prevented them from finding means to establish an increasingly closer personal relationship. The misapplication of the concept of regression resulted in a neglect of the patient's general behavior as well as of personal hygiene and resulted in many psychopathologic aggravations and artefacts. Fortunately, this phase has passed and an individualistic psychotherapeutic approach has resulted in better interpersonal relationships with other patients and with the nursing personnel as well as in attractively furnished wards in hospitals. I remember well my surprise when, in 1925, I saw flowers on these wards in England. My surprise was still greater when I came to the Payne Whitney Psychiatric Clinic in 1936 and found that Dr. William L. Russell had carpets on these floors and ordinary, comfortable furniture. In these modern hospitals, untidiness with urine or feces is rare, table manners are usually acceptable, and clothes are kept relatively tidy. Combativeness does not occur frequently when each occurrence is analyzed critically and the necessary conclusions drawn. There is still too much of a hesitancy to accept these principles fully. It would also help greatly if each hospital had more than one unit for disturbed patients so that separation of potentially combative patients is possible, and a wholesome atmosphere maintained.

A hundred years ago, the controversy of restraint and non-restraint was at its height. Gradually the non-restraint principle became generally accepted but frequently with compromises in its practical applications. From my twenty-five years' experience in psychiatric hospitals, it is my firm conviction that good results can be obtained only if one does not permit any kind of mechanical restraint. It is necessary that such policies be accepted throughout a hospital by all members of the personnel. Of equal importance is the avoidance of isolation by planning for suitable and enforceable temporary separations from other patients. The modern therapeutic aids of insulin and convulsive therapy, of temporary or long-lasting use of sedatives, of occupational and hydrotherapy, and of brain surgery, are of great importance. Indications for the type of treatment and the time when it should be applied have become better understood but still need to be studied further. Whatever treatment is used it must be associaed with well-planned psychodynamic psychotherapy.

From the beginning of the development of modern psychiatric hospitals, for about 150 years, it has been stressed that the head of a psychiatric hospital must be a psychiatrist. The wisdom of such a policy is obvious when one considers the previous discussion. There was, however, a period when hospitals were made so large, that the head of the hospital became merely an administrator and lost the opportunity to remain an active clinician. The unsoundness of such a development was openly stressed in the European countries but only in recent years has there been a change in this country.

The progress of medicine in general had its great influence on psychiatric hospitals. The understanding of nutrition has led to a disappearance of central neuritis, the dreaded end result of semi-starvation. The frequent vitamin deficiency reaction of involutional depressions and psychopathologic disorders of late life are prevented or readily corrected. The application of sound tuberculosis control has demonstrated that tuberculosis has no outstanding affinity to schizophrenia and can be controlled and prevented in the hospital as well as on the outside. I feel justified in making such a broad statement because it has been demonstrated in several hospitals as for example at the Franklin Delano Roosevelt Veterans Hospital at Montrose, New York.

From the beginning work and suitable occupation were used to divert the patients

from their worries and undesirable preoccupations. In some parts of the country there was opposition to the use of menial work for patients of cultured background. Whenever the psychiatrist believed in the therapeutic value of work the opposition to it remained isolated. With a different cultural attitude to work, occupational therapy became generally accepted for all classes of society. During the last thirty years the concept of therapeutic occupation has been changed fundamentally. It became recognized that any kind of occupation is suitable if it is possible for the patient to perform the task well enough to give him some satisfaction in the actual doing, in the contemplation of the goal, and in the achievement of the result obtained. Diversion is still considered important but rather less so than the satisfaction of performing a task adequately. Individual inclinations and interests can be considered in the choice of activities if the psychiatrist is guided by the psychopathologic signs and symptoms. This last point has only been developed recently. While previously depressive retardation or elated distractability was given much consideration it has now been demonstrated that the effects of other emotional reactions may hinder concentration, clarity of thinking, and retentive abilities. Among the most frequent interfering emotions are anxiety and prolonged tension. In addition, resentment and hostility on whatever basis may prevent constructive use of the available time.

Recreational facilities were naturally limited in a society in which only the well-to-do had leisure time. Efforts were guided to suitable outdoor activities. In present society, with interest and active participation in sports, in music and other aesthetic pursuits, and in social games of various kinds, recreation and activities to fill leisure time have become more meaningful and can be adjusted to individual needs. On the other hand, in many hospitals there is a lack of good organization to fill the available time. It is a serious but justified criticism of our modern hospitals that specialists in occupational and recreational activities are guiding the patient for a very short time of the day while most of the time is spent in destructive inactivity. Future treatment will include a well-balanced routine in which suitable work and recreation and adequate time for rest, for meals, and for personal hygiene will fill the whole 24 hours. In such a routine, demands of the hospital environment and of individual spontaneity will be blended. The right for privacy and the obligation to the group will be recognized. Solutions will have to be found for the adequate use of the time when the skilled personnel is not available, e.g., in the evening hours and the still longer weekends. Considerable thought will have to be given to the education of nurses and aides until a satisfactory answer can be found to these problems.

In our schools of nursing and in the training of the graduate nurses insufficient consideration is given to the acquisition of suitable skills in occupation and recreation, based on a sound teaching philosophy. The psychiatrist will have to assume the leadership in the way the surgeon has done in his field. It may well be that some of the answer may be found in a carefully planned and sufficiently varied training program of the young graduate nurse. American medicine has inaugurated the residency program for physicians who wish to select a specialty. Similar principles might be applied to a graduate nursing program, with equal benefits to the individual nurse, the patients and the hospital. It is important that we take the paient out of his usual hospital environment to the occupational and recreational departments but it is equally important that we bring corresponding activities to the wards on which the patient lives and to his bedside. It may be that for some housewife the so-called menial tasks in a hospital are meaningful and satisfactory but, if so, this activity must be well organized and combined with other activities, including social contact and recreation. It may be possible to give a man satisfaction in permitting him to work in an office, in a workship, in a garden or on a farm but it is therapy only if the psychiatrist determines what in these activities is helpful to this individual to achieve self-reliance and adjustment to the demands of the group.

All facilities of the hospital must be reevaluated constantly from the point of view of their therapeutic usefulness or potential-

ities. These principles guide the administrator in the selection of personnel and in their on-the-job training. The atmosphere of a hospital is formed by the whole group, and in some individual cases, the behavior of the lowest employee may be as important as that of the supervisors.

When this hospital was founded American psychiatrists were greatly influenced by the thinking of their French predecessors and contemporaries. There was respect for the dignity of the individual and his rights. This philosophical attitude remained basic in most hospitals. The lack of genuine understanding of the psychopathology of fear and anger, of resistiveness and withdrawal into oneself made it most difficult for the psychiatrists of that period and of several generations to come, to guide the patient skillfully into co-operation and participation in outlined activities. It is therefore not surprising that some psychiatrists felt that obedience had to be enforced and that isolation, withdrawal of privileges, straight jackets, and tranquillizer were considered suitable means to obtain this end. Beneath all this remained, however, the basic philosophy of concern for the individual, and the harsh, but justified, criticism of Dorothy Dix and others was therefore effective. It is an ever-recurring fact in the history of the last hundred years that, corresponding to the medical knowledge of its time, the hospital will be what the public demands and what its conscience tolerates.

A hospital must be part of the life of the region in which it is located. Its obligation to its patients includes an obligation to the members of the patient's family and to the community in which he lives. One cannot merely treat a patient for a given period of time within the walls of the hospital. A physician is concerned with his patient's future, with his later adjustment to life, and with his maintaining as good a state of health as possible. These principles were apparently dimly recognized in the middle of the last century but became stronger and generally accepted as time went on. There is still, however, a long step in most places until the psychiatric hospital becomes the community center of healing of minor and major personality disorders. The concept of intra- versus extramural psychiatry has already disappeared from the thinking of modern psychiatrists. He is as interested in the follow-up observations as his colleagues in medicine and surgery. Ambulatory treatment, whether it be in well-organized outpatient services or in a physician's private office, carried out by psychiatric specialists or by general practitioners, has assumed its important place, closely related and not opposed to hospital treatment. It is recognized that the choice of treatment must be guided by medical indications and contraindications. Treatment in a psychiatric hospital may be merely a phase in a long course of treatment. The hospital offers special opportunities for psychiatric treatment—general as well as for psychotherapy. Being in a psychiatric hospital no longer precludes activities outside the hospital. If local conditions permit, it may be quite feasible to have a patient carry out many, or even most, of his occupational activities in the community while he still resides in the hospital. This type of treatment can be, and is, readily used in progressive therapeutic centers which are located in urban areas. It is more difficult to carry out in rural districts where patients come to the hospital from great distances. Suitable arrangements will have to be found to offer this valuable aspect of treatment to patients in whom it is indicated. Education of the public, of the patient's family, employers and friends is necessary but not as difficult as some seem to think.

It has been over a hundred years since the concept, and the term "mental hygiene" was first introduced. Progress in this field of maintenance and promotion of health of the individual has been slow and has not kept pace with corresponding endeavors in the field of physical health and hygiene. The main reason for this retarded progress seems to me to lie in the fact that psychiatrists, who were trained to detect, study, and treat illnesses applied this same attitude to health. The movement of mental hygiene led to the development of centers for ambulatory treatment. This treatment was directed increasingly to earlier symptoms of illness and attempted to achieve recovery or prevention of further progress of the illness. The under-

lying principle was directed at the correction of maladjustment and not at the maintenance of health.

A person is a biosocial unit. Physical and mental functions are part of biological functioning. This concept has evolved during the last century. The social relations became gradually clearer and their study assumed increasing importance. New methods, fitted to investigate a group of individuals living in its peculiar environment, were developed. These sociological attitudes and methods must be applied if we wish to understand the influence of the social environment on the health of an individual. The anthropologist must help to make one understand cultural influences. The psychiatrist who is trained to analyze the individual and the psychologist with his methods of evaluation of personality traits offer important contributions. No one of these workers alone can solve the problem of health of the personality. The same type of co-operative scientific effort is necessary as in the field of public health.

It is important that we psychiatrists recognize fully the most urgent need to direct some of our thinking and efforts to this problem. Groundwork has already been laid, and steady progress can be expected. It is most fitting that the contribution of Tennessee be mentioned. The work of Drs. Luton and Roth represent an important milestone, which was necessary for the development of social psychiatry which was started at Cornell University Medical College a year ago. The study of rural and urban districts alike, of Tennessee and of New York City, are necessary correlates for an understanding of the health of our population.

The psychiatric hospital of the future will become the hospital for all types of personality disorders and for all ages. The present artificial limitations will gradually disappear. The chronic alcoholic and the drug addict will be admitted for treatment as well as the psychoneurotic patient. When scientifically unjustifiable separation of the neu- follow a medically sound policy for admission procedure. It will be found that a small percentage of patients will have to be committed by law and be kept against their will. A psychiatrist who tries to utilize the patient's feeling of illness and avoids trying to reach him through reasoning and so-called insight will be able to establish and maintain a helpful relationship with most of his patients. Reviewing the patient's illness with the distressed and frequently frightened and bewildered relatives will make it possible to gain their co-operation. At the Payne Whitney Psychiatric Clinic I have always avoided forcibly retaining a patient although the hospital has this power. Whenever the above outlined therapeutic procedures were followed and the help of the responsible relatives obtained, the patient was either willing to stay in the hospital or able to accept the necessity of hospital treatment. The patients who had to be discharged against advice were severely damaged psychoneurotic or psychopathic individuals. In other words, the patients who would be admitted to the average psychiatric hospital and usually under legal commitment are those who are willing to stay for treatment. When the public has accepted the fact that a psychiatric hospital is not a place for detention, the patients will be willing to seek help.

The progress in medical treatment in general and the availability of modern laboratories make it possible to treat patients suffering from physical complications. Conditions in which the personality maladjustment is expressed in physical symptoms, the so-called psychosomatic disorders, may sometimes be best treated in a psychiatric hospital although the vast majority should be treated in general hospitals and in ambulatory practice. In order to achieve the highest type of treatment of physical complications a psychiatric hospital can no longer afford the medical self-sufficiency which has been characteristic in the past. It will be necessary to bring in the best consultants for a given problem and to establish affiliations which will be mutually profitable.

Every large psychiatric hospital takes care of acute and chronic conditions. Among the latter group are patients who may spend many years, even from youth to old age, in the hospital. In order to practice good medicine a physician must examine these patients physically each year. It seems justifiable to ask whether these examinations

should be carried out by psychiatrists instead of general practitioners as it is done in every other community. The psychiatrist is a physician who is highly trained in the study of psychopathology and in the intricate aspects of psychotherapy and many other forms of psychiatric treatment. A sound basic knowledge of physiology and internal medicine is essential and must be kept alive. The physical condition must be taken into account at all times in every patient. There is, however, a great step between the study and treatment of a psychiatric disorder, with full attention to all possible physical implications and complications and the health examination of a supposedly physically healthy person. It would seem that a basic reorientation in the use of medical personnel is needed. We cannot afford to give the highly trained specialists, whether they be surgeons, psychiatrists, or internists, tasks to perform in which their special skills are not fully utilized.

A psychiatric hospital is a large community with manifold health problems and a variety of illnesses. It is in the nature of this hospital that personality disorders present the dominant medical problems. In addition, there are numerous other medical problems as found in any large community, including serious and minor respiratory, cardiovascular, and gastrointestinal disorders, minor injuries as well as surgical illnesses. Physicians need to attend to these ailments but there is no community which would feel justified in expecting specialists to fill these functions.

At the Franklin Delano Roosevelt Veterans Hospital at Montrose, New York, a study is in progress to determine the respective medical functions to be carried out by general practitioners, internists, surgeons, and psychiatrists. In the present exploratory phase of this study, a general practitioner, that is, a well-trained physician who has not had specialized training makes rounds through the buildings assigned to him accompanied by the psychiatrist. In this way, it can be shortly determined what patients need his attention. In addition, he carries out the annual examination of patients and is responsible for this part of the record and the necessary reports. Whenever it seems indicated he will ask for consultations from the respective specialists. Being assigned to the department of internal medicine in this hospital prevents this physician from becoming isolated or from becoming bored through so-called routine work. It seems to me his work is just as interesting as it would be in his office or in his visits to a bed patient in any other community.

In the field of nursing a similar type of reorientation has been started. The modern nurse carries out many tasks which were previously considered the obligation of the physician. If we want to utilize the constantly increasing knowledge and skill of the nurses to the utmost, we cannot afford to let their time be taken up with tasks which can be delegated to other personnel. It is not a luxury to have a well-trained clerical force available in a hospital which will be responsible for certain routine activities, but it represents a considerable saving because it permits the highest paid professional group to concentrate on problems for which they have been most carefully trained.

In recent years the psychologist has assumed his important place in the hospital. In the field of personality testing the psychologist offers a service which corresponds to the other service departments of a hospital, e.g., clinical pathological laboratory or x-ray department. In order to use such a diagnostic laboratory to its best advantage all the physicians must be acquainted with the value of the tests performed, their reliability and their validity. It is a sign of inadequate medical knowledge and practice if a physician asks in a general way for a large number of tests instead of tests which seem specifically indicated for the medical problem under consideration. In order to live up to the highest medical standards the hospital authorities should expect the physicians to understand the basic principle of the tests used, their mode of application and what they can be expected to reveal. If a physician knows the indications for tests, they will be applied critically and much time and effort saved. It might be well if the hospital administration would acquaint physicians with the expense involved in giving tests whether it be in the time consumed or in the amount of dollars spent for salaries. In many

general hospitals this procedure has been followed and resulted in better medical practice and especially in better mutual understanding between administrative and professional groups.

The role of the psychologists might well be expanded further but we deal here with a field which has not been sufficiently explored. We cannot delegate to the non-psychiatric staff, whether they be internists, general practitioners, or psychologists, the tasks of psychiatric treatment. It would be an undesirable and frequently disastrous step if one would, as it has been proposed, put non-psychiatrists in charge of chronic psychiatric patients. All these patients demand treatment along psychiatric lines. Whenever treatment of these patients is replaced by taking care of them, medical standards will deteriorate and the patients will suffer from psychiatric neglect. It should become possible to integrate the psychologist in the psychiatric treatment of a group. Such an integration is, however, not feasible if the psychologist attends in the limited span from 9 A.M. to 5 P.M. and is not acquainted fully with the behavior and the activities of patients during 24 hours.

There has been a changing pattern of the type of psychiatric illnesses admitted to hospitals. General paresis has decreased greatly and senile and other aging disorders begin to fill the hospitals. Thought must be given to this problem in the future construction and utilization of our hospitals. It seems to me more important to find solutions in the communities. Many senile patients could be taken care of at home if suitable conditions existed. It might well be that a similar system could be developed for old people as has been found helpful for small children who are put in nursery schools while the mother works. A large number of confused elderly patients and others who suffer from marked memory disorders and beginning habit deterioration could be left by the responsible relatives in a suitable outpatient unit when they are on their way to work. In such a well organized unit with planned activity, rest and light meal periods, and with some medical supervision, patients could pass the day constructively and contentedly. The evenings, nights, and weekends would be spent with their families and in their accustomed home. Most daughters and sons dread to send their parents to a hospital for their declining years. They would be glad to spend some of their earnings to have them cared for.

The principle of housing psychiatric patients with private families or in small, self-sustaining colonies has never taken firm root in this country. Even a hundred years ago American psychiatrists noted with interest the old family care program of the Belgian town of Gheel. This interest still exists but has never born fruitful results. There is no question that this type of psychiatric treatment deserves attention. It will be necessary to study the whole problem from a fresh and modern point of view.

To have a patient return to his community or even to a life outside of a hospital may not be the most desirable goal of treatment. It may be best for some patients to remain the rest of their lives in a hospital which offers a true asylum for them, a place in which they can function to the best of their abilities. Others may need treatment in a place to which they can retreat temporarily from the problems of their lives. A physician must guard against making his philosophy of life the one for his patient and expect him to find satisfaction in situations which will always remain intolerable to him. To keep a patient outside of a hospital or to have him return to life outside may be false goals.

Progress in hospital treatment has been considerable in past years and will continue uninterruptedly in the future. Its course and its speed will depend on the progress of medicine in general, on our cultural developments in their broadest sense, and on current philosophies and attitudes to life. These are the factors which will form the standards of treatment. The solution will, however, have to remain adjusted to local needs and be worked out by the leaders of the hospital and of the community which it serves.

Variation of E. E. G. Patterns in a Case of Neurosyphilis

LESLIE J. SEELEY, M.D., and RODGER S. DILLE, M.D.

American Lake, Washington

A case of neurosyphilis is being presented in which the E.E.G. at the onset of seizures was normal, became abnormal between courses of penicillin therapy and reverted to normal following treatment. The abnormalities of the electroencephalogram in neurosyphilis and the improvement following therapy have been the subject of numerous reports.[1]

There have been reports of abnormal brain waves due to neurosyphilis[1] and other diseases, such as encephalitis. Gibbs[2] cites the etiological factors in 240 cases of encephalitis, the main ones being influenza, measles, whooping cough, pneumonia, scarlet fever and others of unknown origin. Greenblatt and Levin[1] report the improvement in brain rhythm of a number of cases of neurosyphilis following chemotherapy and fever therapy.

Scott[3] reported the marked improvement in E.E.G. records of almost all cases of neurosyphilis following penicillin therapy. Many observers[4] agree that the clinical improvement does not always parallel the E.E.G. improvement.

It is believed that this case is unique in that a normal E.E.G. was obtained in the initial stages of the neurological manifestations, became abnormal, and later reverted to normal after treatment and with improvement in symptoms.

A 35 year old white male (W.F.C.) developed a primary penile lesion in July, 1939. Inadequate treatment (6 intravenous and 12 intramuscular metallic injections) followed. Presumably his serology was negative on joining the armed forces in April, 1941, but following his hospitalization for hernioplasty in an Army hospital in 1942 the Wassermann was found to be 4+ and Kahn 3+. His spinal fluid Wassermann and gold curve were positive. He was given another short course of metallic injections and sent overseas. The record of his luetic infection did not catch up with him until 1944. At this time his blood and spinal fluid was found to be positive and he was returned to the United States and subsequently discharged from the service in December, 1944.

He was first admitted to Veterans Administration Hospital, American Lake, Washington, February 2, 1948 because of an atypical convulsive seizure, was released from the hospital on his own request March 17, 1948 without treatment or serology report. The second admission, August, 1949, followed an atypical convulsive seizure and mental confusion. A general and neurological examination was negative. Spinal fluid findings, Wasserman 3+, gold curve 1100000000, blood Wassermann 4+, blood Kahn 2+. An initial E.E.G. August 28, 1949 showed a normal pattern with basic rhythm of 9/second (Fig. 1). This record was made on a 4-channel machine, using Gibbs' technic.[5] He was discharged after a course of 9,000,000 units penicillin, with instruction to return in three months for a check-up. He failed to return.

A third admission in September, 1950, again followed an atypical convulsive seizure. A general and neurological examination was again negative except for sluggish pupils. An E.E.G. on October 9, 1950 (Fig. II) showed a basic rhythm of 9/second with paroxysmal high voltage, slow waves (1½ to 3/second) in all left anterior leads with some slight spread to the right anterior temporal, temporal, frontal and parietal regions. No seizure activity during sleep, no change with hyperventilation or following metrazol activation (10 cc of 5% metrazol I.V. at 2 cc/minute). This record was made on a 6-channel model, using Gibbs' technic; monopolar leads, reference electrodes at times to both ears, single ear, and other indifferent reference points. An electroencephalogram was normal. A second course of penicillin (4,515,000 units) was given in

From the Veterans Administration Hospital, American Lake, Washington.

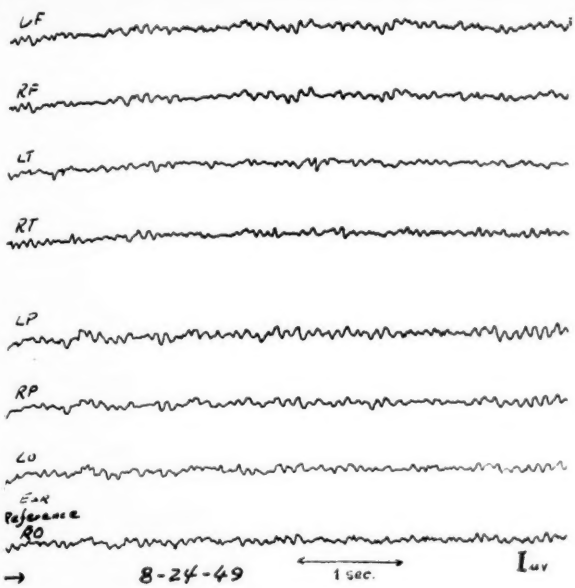

Fig. 1. Basic rhythm 9/sec. No seizure activity. No amplitude asymmetry. No bursts of slow waves. No change with hyperventilation. Normal E.E.G., awake and asleep.

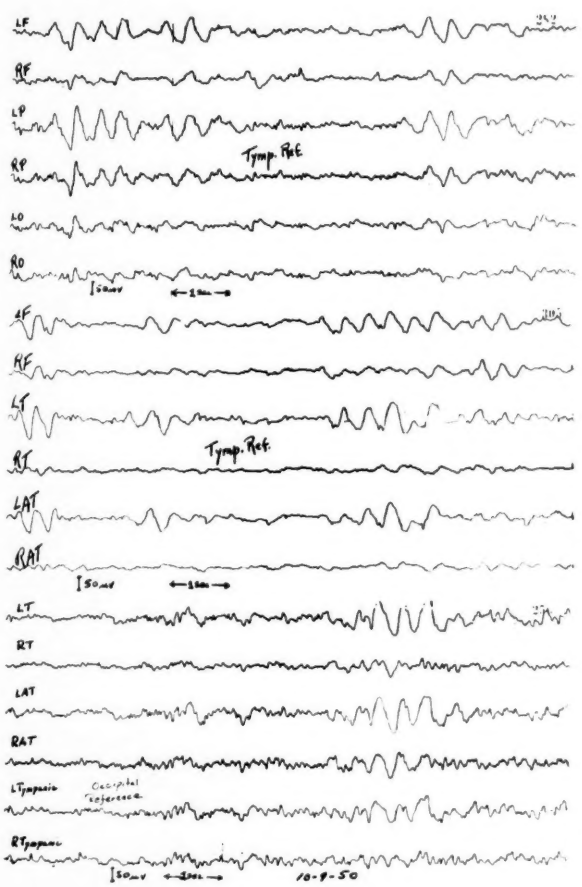

Fig. 2. Grossly abnormal E.E.G., with high voltage, paroxysmal, slow waves (1½-3/sec.) in entire left anterior cerebral cortex, and some spread to the right parietal and right anterior temporal leads.

October 1950. The blood and spinal fluid remained positive and he was given a third course (9,015,000 units penicillin) in November 1951.

A third E.E.G. recording made in January 1952, two months after his third course of penicillin (Fig. III) revealed a return to normal cortical rhythm. The interpretation of this record is as follows: Basic rhythm 9/second, no seizure discharges, no build-up with hyperventilation. Absolutely no abnormal findings in the entire record. From this it is apparent that the gross brain damage, evident 15 months before, had disappeared. A fourth E.E.G. was recorded in August 1952 which was entirely normal and quite similar to E.E.G. I and E.E.G. III.

Gibbs[2] points out that in many cases of encephalitis the E.E.G. is characterized by slow waves of high voltage in the acute stage. These slow waves may be generalized throughout the cortex, or may be localized. In cases of encephalitis without seizures, the E.E.G. tended to return to normal in the post-acute phase of the disease. In 58 percent of those having seizures due to

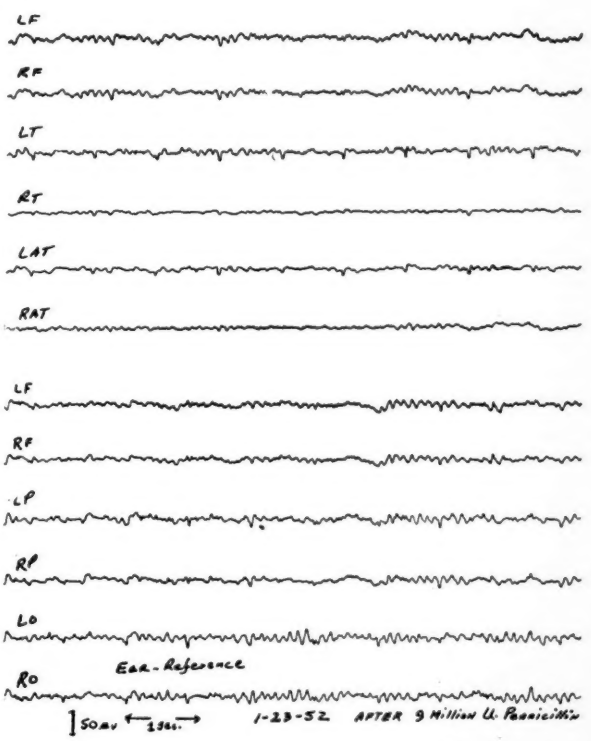

Fig. 3. Basic rhythm 9/sec. Much E.K.G. in the left temporal leads. No slow wave activity. No amplitude asymmetry. No seizure discharges. No change with hyperventilation. Normal E.E.G., awake and asleep.

encephalitis the E.E.G. revealed paroxysmal seizure discharges in the post-encephalitic phase.

From the case under discussion it is evident that the E.E.G. recordings in patients with seizures due to neurosyphilis (paresis or menigo-vascular syphilis)[1] differ radically from those due to encephalitis in the post-acute stage.

Discussion

The exact nature of reversible lesions present in central nervous system syphilis which would explain the changes observed in this case are unknown. One may speculate either that the leptomeningeal involvement which is present so universally in all forms of CNS lues has altered the function of the underlying cortex, or more probably that some reversible localized endarteritis of the fine cortical vessels was present to produce the observed changes (6, 7 & 8). Syphilitic endarteritis has always been relatively rare and it is obvious that in the present antibiotic era of syphilotherapy will be even more rare.

The well known tendency for luetic endarteritis to localize in various parts of the brain,[6,7,8] especially the cortex, and the variability of the symptoms, as compared to the microanatomical findings would well account for these focal (left sided) E.E.G. findings with but negative objective neurological examinations. The seizures, of course, are explained adequately by this cortical involvement. Thus syphilitic endarteritis is a pathological diagnosis and cannot be ascertained clinically with certainty, the effects of therapy have been only speculative until the advent and improvement of E.E.G. The known histological picture of this disorder, however, does not preclude reversible possibilities in the early stages of the process.

There are but few reports, none of recent date, concerning the E.E.G. findings in various types of neurosyphilis. Greenblatt and Levin[1] reported that 91 percent of patients with neurosyphilis and seizures had E.E.G. abnormalities, 23 percent of which had paroxysmal patterns. Forty percent of CNS lues without seizures had abnormal E.E.G. findings but only 1 percent of these had paroxysmal types. It is of interest to contrast these figures with those of Gibbs and Gibbs[2] who showed that 87 percent of post-encephalitics from infections other than syphilis, with seizures, had abnormal E.E.G. findings, of which 58 percent had paroxysmal patterns. Also 18 percent post-encephalitics without seizures had abnormal patterns, but again only 1 percent were of the paroxysmal type. Bennett[4] and Finley showed that the E.E.G. in neurosyphilis improved with various forms of fever therapy and chemotherapy. Bennett[4] in discussion remarks that the slow potentials seem to be an indication of an acute process without parenchymal destruction, because he found in a series of cases a return to normal E.E.G. patterns after treatment. He also noted a tendency to asymmetry of the slow paroxysmal type in 4 percent of the cases studied. It should be kept in mind that electroencephalography has made great strides in the past ten years (when Bennett made these observations), both in technic and in interpretive experience.

Summary

A case of neurosyphilis has been presented in which a normal E.E.G. pattern was found at the onset of seizures. Without treatment during a one year interval, the abnormal E.E.G. pattern developed. Two months following penicillin therapy the pattern had reverted to normal and has continued to remain normal during the past seven months.

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Rapid Coma Technique of Carbon Dioxide Inhalation Therapy

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INTRODUCTION: Single breaths have been used by many workers in the field of Carbon Dioxide Inhalation Therapy in order to habituate the patient to the multiple breath treatment of 20 or 30 per cent CO_2 and oxygen mixtures. However, the technique herein described consists solely of forced inhalation of the single breath of a much stronger concentration of CO_2 (70 per cent CO_2 and 30 per cent oxygen) preceded by a forced exhalation of room air, producing a rapid coma of brief duration.

HISTORY OF DEVELOPMENT: While searching for a way to overcome the anxiety and the fear associated with the multiple breath technique of carbon dioxide inhalation therapy, I began to have the patient take a single breath of 30 per cent CO_2—70 per cent oxygen mixture with a forced inhalation, preceded by a previously forced exhalation of room air. In so doing the patient described a sensation of almost going under. By encouraging the patient to retain the inhaled mixture, a still deeper stage of relative unconsciousness was reached. However, the patient still complained of "nervous tension" in the viscera and musculature. By increasing the concentration of the CO_2, it was relatively simple for the patient to become momentarily unconscious with a single deep breath, without residual tension in the body. In experimenting with the various concentrations I found that a mixture of 70 per cent CO_2 and 30 per cent oxygen gave good results.

No toxic or other serious irreversible effects of this treatment utilizing the heavy concentration of CO_2 have been noted in over 4,000 single breath treatments administered to a large number of patients. This technique, therefore, appears to be safe.

This paper was presented at the Symposium on Carbon Dioxide Research and Treatment held at Atlantic City, New Jersey, May 12th, 1952.

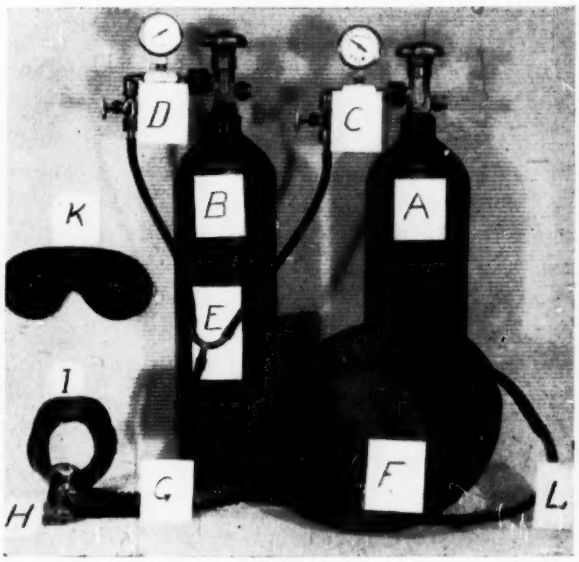

Figure 1.

A. Cylinder of 100% Carbon Dioxide (with flow meter).
B. Cylinder of 100% Oxygen (with flow meter).
C. & D. Reducing valves with manometer to indicate internal pressure of cylinder and needle shut-off valve attached.
E. "Y" Tube.
F. 11 litre breathing bag (filled).
G. Corrugated rubber extension.
H. Chimney piece with three way valve.
I. Large face mask (dental anesthesia type).
K. Sleep shade.
L. Rubber tubing (5/16").

DESCRIPTION OF TECHNIQUE:

Equipment and Apparatus

Each cylinder is fitted with a flow meter of the standard variety which indicates flow per unit time, and connected through a "Y" tube into an 11 liter breathing bag, at the end of which is an anesthesia mask fitted with a shut-off valve which permits filling of the breathing bag, and discharging its contents into the face mask when opened, and also permitting the breathing of room air when shut off, while the mask is applied to the face. Since pure mixture of gases is used, it is relatively simple to compute the 70 per cent CO_2 30 per cent oxygen mixture

to fill the breathing bag for each single breath treatment. The mask applied to the patient's face is preferably a large dental anesthesia mask, made of plastic, which can be comfortably applied to the face with the mouth *wide open* and with the nose within the applied mask. A sleeping shade, which covers the eyes, prevents light from entering the closed lids. (This is used in order to accentuate the visualized dream pattern which appears in the short space of treatment and is optional.)

Method:

The patient is placed in a supine position on a bed or stretcher and instructed, with practice, how to exhale and inhale room air, forcefully and maximally, with the mouth as *wide open* as is possible, with the breathing mask gently and comfortably on the face. He is told that when he has inhaled his absolutely maximal breath of the gas mixture, to signal the doctor by raising his hand, and that the mask will immediately be withdrawn from his face. After a single deep maximal exhalation of his own breath the valve supplying the gas to the mask is turned on and the patient encouraged to breathe a single deep maximal inhalation of the gas mixture with his mouth fully opened. There is the usual leakage of gas and room air about the periphery of the mask; positive pressure to prevent leakage of room air into the mask is created by firm but gentle pressure exerted by the doctor with the hand against the breathing bag in order to assure adequate and constant gas inhalation by the patient at all times. When the patient gives the signal that he cannot inhale any more gas, the mask is quickly removed. At this time the patient is still fully conscious. He then rapidly enters a carbon dioxide coma, or subcoma, or hypnagogic state, during which he almost invariably visualizes a geometric, colorful pattern with or without formed symbols. Within a matter of eight seconds he is fully awake and recalls every detail of the dream. In the meantime, he demonstrates autonomic and motor body reactions consisting of skin changes, perspiration and flushing, mottling, motor activity and vocalizations, but without the usual severe exhaustion that follows the multiple breath technique of prolonged carbon dioxide (20 to 50 breaths of 30 per cent CO_2—70 per cent oxygen mixture). Hyperventilation in the meantime has occurred, as with multiple breath technique, and the CO_2 mixture is quickly blown off.

Number and Frequency of Treatments:

This is an individual problem to be determined by the physician. Usually a three-time per week schedule in which one to ten single breath treatments are administered with each session is sufficient. An interval of at least three minutes (so that pulse and blood pressure return to normal) is used between single breath treatments. Psychotherapy may be utilized between treatments, if the physician so desires. If the above mentioned schedule does not produce improvements in the patient clinically, after three to four weeks, then intensive treatment is required, which consists of giving the patient up to 20 single breath treatments daily for a period of two weeks. During the course of treatment the patient frequently feels stimulated, manifesting increased psychomotor energy and signs of stimulation, but no unusually unpleasant symptoms. After the treatment series is completed, the patient may experience fatigue, mild symptoms of exhaustion, mildly increased irritability, and a slight increase of intensity of the subjective symptoms for a period of 48 hours. Thereafter, he rapidly reverts toward normal function with a sense of well-being and a new emotional stability, which he frequently describes as a "feeling of calm and serenity."

Occasionally patients who are very anxious will make better progress if they are permitted to apply their own treatment, such as holding the mask, and turning the valve on by themselves. In these patients combinations of methods may be used in which the physician may give the treatment with the patient signalling, alternated with the application of the treatment by the patient alone.

Contra-indications:

Similar to those described by L. J. Meduna, M.D., in the multiple breath technique, namely, severe hypertension, pulmonary tuberculosis, coronary heart disease, severe emphysema.

ADVANTAGES OF THE RAPID COMA TECHNIQUE:

Psychological Advantages:

Minimization of the psychologic fears usually associated with Carbon Dioxide Inhalation Therapy of the multiple breath technique:

(a) A fear of suffocation or air hunger sensation, which is frequently encountered in the induction phase of the multiple breath technique.

(b) Apprehension of the day of treatment.

(c) Anxiety that develops during the course of carbon dioxide inhalation therapy of the multiple breath technique (from 10 to 20 treatment).

Physiological Advantages:

There is less physical exhaustion with the rapid coma technique. It is physiologically impossible to raise the overall concentration of CO_2 in the blood to the toxic or harmful levels, since only a single breath is inhaled into the lungs, and only part of it is absorbed into the pulmonary circulation, then rapidly diluted in the large blood volume of the body fluids, and quickly blown off via the lungs when the mask is removed.

Technical Advantages:

Greater economy of gas consumption.

DISADVANTAGES OF THE RAPID COMA TECHNIQUE:

Commercially mixed gases of the 70 per cent CO_2—30 per cent oxygen mixture are not available in a single tank.

The greater complexity in filling the breathing bag with the proper mixture, since there may be errors of five to ten per cent—(however, such range of errors is not serious or harmful, provided there is at least 20 per cent oxygen in the breathing).

Difficulty in breathing a single breath of the concentrated mixture of 70 per cent CO_2. This occurs in 10 per cent of the patients, and this is due to irritative effect of the gas producing a reflex arrest of the respiratory motion, over which the patient has no conscious control as long as the mask is on the face and the valve is turned on. This reflex arrest may be physiological or psychological, but in either case it is readily overcome by starting the patient to breathe single breaths with a lower concentration of CO_2 and gradually increasing to 70 per cent CO_2.

MECHANISMS OF ACTION AND RATIONALE OF THE RAPID COMA TECHNIQUE (70 per cent CO_2—30 per cent Oxygen):

Speculative considerations: Because of the much quicker saturation and the higher instant concentration of the CO_2 in the blood and central nervous system, there is less opportunity for the formation of adaptive resistances in the tissues and cells.

Experimental and theoretical evidence of the rationale of Carbon Dioxide Inhalation Therapy of the multiple breath technique has been extensively described by Loevenhart, et al., L. J. Meduna, C. F. Schmidt.

RESULTS: The Rapid Coma Technique has not been used sufficiently to permit an adequate evaluation of its long-range therapeutic effectiveness. However, a substantial number of patients previously treated with the standard multiple breath technique (30 per cent CO_2—70 per cent oxygen mixture) without notable clinical benefit manifested significant improvement when treatment with the Rapid Coma Technique was subsequently applied. Furthermore, a number of patients who have thus far been treated solely with the Rapid Coma Technique appeared to have shown a favorable clinical response.

CONCLUSIONS:

1. It is emphasized that follow-up studies in the above cases treated with the Rapid Coma Technique have not been of sufficient duration to warrant definite conclusions.

2. It is suggested that this new technique be utilized by other investigators in order to compile additional data for more valid evaluation.

Discussion

Dr. Payl H. Wilcox, Traverse City, Michigan: I would like to explain the problem of mixing carbon dioxide and oxygen. The reason one cannot get more than 30 per cent carbon dioxide into an oxygen tank is because a higher percentage of carbon dioxide under high pressure, like 1800 lbs. per square inch, liquifies. The liquified carbon dioxide may solidify in the tank in the form of dry ice. If the pressure decreases in the tank due to using the gas,

more and more of the liquified and frozen dioxide turns again to gas and therefore the mixture will be unreliable, but if one produces the mixture at a low pressure as Dr. LaVerne did in the bag, or if one uses an anesthetic machine with an ordinary mixing valve, one can set the valve and get any mixture from zero to 100 per cent.

Dr. Claude Uhler, Dallas, Texas: Is there any advantage in pressing the inflated bag? I noticed that the bag was inflated beyond that of the pressure within the lungs. Is there any advantage in filling the bag to the point so that the gas is forced into the lungs?

Dr. La Verne: We can produce a mixture of 40 per cent carbon dioxide and 60 per cent oxygen in a tank under high pressure, but none higher, for the reasons mentioned by Dr. Wilcox. As to Dr. Uhler's question, I can answer this: When the mouth is open the patient likes to feel that gas is available and they want it under pressure. Secondly, there is always leakage around the mask, and since the patients are sucking the gas in deeply if there is not positive pressure in the bag they will suck in room air and will not go into a coma.

Question: In treating patients I have found that men with large chests will frequently empty a 5 liter bag with one respiration without exerting themselves, and I do not know if this technique would satisfy their demand for the gas.

Dr. La Verne: This is the reason I use an 11 liter bag.

Reactions of Patients to Textbook-Mediated Group Psychotherapy

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Textbook-mediated group psychotherapy quite naturally falls into the category of the didactic. In a sense, it may even be considered the apogee of didacticism, mediated as it is so largely through a book. Its special advantage lies in the fact that it more readily engages the residual capacities of more poverty-stricken personalities in which free association or any spontaneous associations prove to be beyond the subjects' capacities, or are at least most labored. As a source of stimulus material its effectiveness with better-endowed individuals is by no means excluded. Besides the advantages in systematization of stimulus material, the use of the textbook helps to replete personalities drained of content and intellectual curiosity, and thus permits reconditioning to the point where greater exploration of character and personality is made possible. The technique of textbook-mediated group psychotherapy has been explained elsewhere.[1]

The point is well illustrated by a group of post-lobotomy patients. Believing that such a group, consisting mostly of regressed and deteriorated schizophrenias, would offer no opportunity whatever for a textbook-mediated form of psychotherapy, the therapist, meeting with the group for the first time, intended merely to give some explanatory remarks and indicate some of the objectives. But a sizeable proportion of the group sat silently slumped over in their chairs, entirely oblivious to their surroundings. Others talked aloud at random. At the same time, in shifting locales, one turbulence collided with another and fisticuffs and hair-pulling seemed on the verge of breaking out. No sooner had the therapist quieted a disturbance in one part of the room than another began in a new quarter. Therapist might begin to address this crew about the purposes and intent of the forthcoming meetings and accomplish a sentence or two when a raucous argument would break out between two lobotomees, or one of them would begin a loud declamation, at the same time that another or several would break out in peals of autistic, apparently unmotivated laughter. Thus therapist could not get very far with his explanations.

Discouraged with the enterprise, therapist was about ready to give up the project. One thing seemed clear. These shorn lambs would never do for textbook-mediated group psychotherapy. Not only was there insufficient residual emotional and intellectual capacity, but for other reasons, too, they seemed to require a far more rudimentary approach if any form of psychotherapy were

to be administered. As a consequence, for the second session therapist groomed himself to deliver a very simple address, attempting to avoid any involved concepts and to employ the simplest of diction.

Thus forearmed he came to the second class period and began his simple discourse. Then the same thing began to happen as on the first session. Their attention could not be mobilized, and therapist's eloquence died a-borning. He could scarcely get through a sentence before several jabberwockies would start arguing and he would have to stop his discourse and attempt to restore order. It would be a foolhardy orator indeed, who would hope to spellbind these mavericks into a semblance of a cohesive, smoothly functioning group psychotherapy class. Therapist concluded he was not equal to the required oratory.

The textbook[2] therapist had been using with his previous groups did not seem suitable for the kind of regressed patients composing this group. Out of desperation he decided to bring the books for the third session and if that offered no possibility of continuing with the group he would relinquish the project. With no great enthusiasm he brought the books to the third session. Passed around, a book to each patient, there was still much disturbance—the loud declamations, the peals of autistic laughter. But patients began turning pages with what seemed like supercilious disdain.

However, when called upon to read aloud they complied, and after several such sessions there was a great reduction in class disturbance, e.g. the vacuous declamations, the pointless squabbles, the torrents of autistic laughter. General attention was at least 100 per cent improved. Three patients handed in "homework" quite regularly for a time. The reading aloud from the textbooks seems much more intelligible and there has been considerable spontaneous comment. A number of the original group has sufficiently improved to be discharged and at this writing are out of the hospital. Most of the remaining patients and those since added to the class, by reports of ward personnel, are showing distinctly improved ward behavior and adjustment.

In these lobotomees it is not difficult to distinguish general levels of integration, or degrees of residual personality organization. It is more than possible such categories can be more or less objectively and accurately established by batteries of psychological tests, but they are not too difficult to detect grossly from observation with the aid of the "naked eye."

Such criteria will delineate three general strata of these patients. We may call them "top-flight," "middle-flight" and "bottom-flight" patients. A questionnaire was given to these patients, about 25 in number; 19 were filled out and returned in various grades of completion. The questions were:

1. What part of this classwork have you found most interesting and helpful.

2. What part have you found least interesting and helpful.

3. Have you understood what was being talked about during the group sessions?

(a) Most of it . . .

(b) Some of it . . .

(c) Not at all . . .

4. What topics have you found of greatest value? Explain why.

5. What topics would you suggest should be discussed in the group?

6. Did anything discussed in the group help you to understand yourself a little better? How? Explain fully, if necessary using an extra sheet of paper here and numbering it to correspond with this question.

7. Did anything discussed in class help you to get along a little better with other people, family, friends, etc.? What topics helped you in this respect? How did it? Describe fully, using an extra sheet if necessary and numbering accordingly.

8. What have you thought about the "Homework"? If you wish state why you prefer not to do the "homework." If you do the "homework" explain if you think it helps and in what way. Discuss fully, using an extra sheet of paper if necessary.

9. Do you feel you still need help in your personal problems? What kind of help? Discuss fully. Use an extra sheet of paper if necessary.

10. Do you like the idea of using textbooks in the class? State your reasons, whether for or against. Explain as fully as you can, using extra paper if necessary.

The answers to 2 of the questions, 3 and 10, given by these lobotomees are listed herewith.

"BOTTOM-FLIGHT": TOTAL 9 PATIENTS

Question 3

(a) 3
(b) 1

One patient abstaining from answering this question, and two checking all the subheads.

"MIDDLE-FLIGHT": TOTAL 7 PATIENTS

(a) 3
(b) 3
(c) 1

"TOP-FLIGHT": TOTAL 3 PATIENTS

(a) 3

Question 10

"BOTTOM-FLIGHT"

Patient No. 1 No answer.
" " 2 " "
" " 3 " "
" " 4 "
" " 5 "Yes."
" " 6 "Yes."
" " 7 "Yes I like the idea of using the textbook; yes I do; depending on the subject explained."
" " 8 "Yes, yes, yes—because we, exercise our eyes—well maybe my husband might come some day, to—Sure—As long as you too are *ok*—but don't let us down. Thank you. three lines extra any (D. I miss my sister)."
" " 9 "Yes, I like extra textbooks. Yet by using state textbooks in class whether for or against or not."

"MIDDLE-FLIGHT"

" " 10 "Using textbook is very helpful especially when you discuss it to some extent."
" " 11 No answer.
" " 12 No answer.
" " 13 "Yes."
" " 14 "Yes I do like the idea of using textbooks in class. The reason is it makes things clearer and more explanatory."
" " 15 No answer.
" " 16 "Yes—I like the idea of using textbooks in class—Getting what is being discussed is much easier by all reading a textbook."

"TOP-FLIGHT"

" " 17 "Yes. Using a textbook in class makes it easier to understand the subject that is being discussed."
" " 18 "I do approve of having textbooks in use, because they help us follow a definite outline."
" " 19 "I think I would like using textbooks in class. I would be able to learn more about different subjects and give me a chance to see if I am able to be helped as much as I should."

It must be explained for the sake of accuracy in evaluation, that statements of the more regressed patients are of variable validity. In the more severe grades of regression there is often present a kind of vacuous pliability in which a "yes" or "no" answer to the same question can be elicited with almost equal facility—and yet the benefit of the doubt can still safely be accorded them. As the scale is ascended toward the "top-flight" members the degree of reliability of responses may be presumed to increase correspondingly.

Thus with better preserved groups of nonlobotomy psychotic patients treated in an out-patient clinic the answers to the same questionnaire may safely be assumed to carry a high degree of validity as frank statements of feeling and conviction in the matter.

Out of a group of 15 patients 9 questionnaires were completed. The answers to two of these questions are listed below. To the question, "What part of the classwork have you found most interesting and helpful?" the answers were:

Patient 1. "Primitive man, his customs and development, tribal customs, the medicine man, and early concepts of disease. Progress in treatment and care of psychosis. 'Frozen' instinctual patterns; adaptation. Word origins, meanings, semantics."

Patient 2. "I truly think all of it was interesting and helpful, and mainly the portion which seemed to help me see myself as others see me."

Patient 3. "The discussion of psychoses and explanation of some of their effects such as 'projection'—that is, misinterpretation in perception caused by the troubles that lie within the mentally ill."

Patient 4. "To hear someone summarize a paragraph, because I don't get the meaning of it, unless I read it over several times."

Patient 5. "I have found all the classwork interesting. I learned to talk without being afraid that I will say the wrong thing. I can read aloud and it has meaning, and lately I have found that I can picture things and words have become more meaning."

Patient 6. "The discussions which pertain to my case and help me to understand better."

Patient 7. "When we discussed the stigma of being in a mental hospital; also studying about the mind."

Patient 8. "Meeting other patients and studying about emotions has helped me most."

Patient 9. "I have found the lectures themselves most interesting (commentaries and amplifications of the text by therapist)."

To the question: "Have you understood what is being talked about in the class sessions?" the answers were:

(a) Most of it	6
(b) Some of it	3
(c) None of it	0

Especially noteworthy were the comments of patient No. 1 in answer to another question. He was an intelligent, seclusive young man, a lawyer by profession, who had suffered a schizophrenic breakdown and had been hospitalized and received electroshock therapy. At this time he was on out-patient status at the clinic. His father had been a well-known psychiatrist.

"Paragraph restatement of the text, it seems to me, is not conducive to continued or unflagging interest. Objections to the method are:

"(a) Participation is momentary and largely applies only to the person reciting.

"(b) The method is unconsciously resented as juvenile.

"(c) Emphasis is on words rather than ideas and concepts.

"(d) Encourages tension as feat of memory.

"Most of these objections are overcome by such questions as, 'What do you think of this paragraph?', 'Do you agree?', 'Why do you disagree?' Answers to these questions stimulate receptivity. The subject-matter and its retention, instead of being an end in itself, and hence subject to mental inertia, tension, resentment, etc., becomes easily assumed background and direction-indicator for enlargement, outlet for expression and feeling in comment. Hence the amount of text material covered is less important than the completest and most spontaneous class use and development of the specific material covered.

"People called in turn are apt to become inattentive until approach of the next call on their performance. The scattering of questions helps to maintain attention. Classes have been extremely well moderated, and assuming the continuation of this control, the best sessions seemed to be marked by the freest, most open discussions. Espousal and contention are contagious, and controlled, help to generate and mold morale."

As a result of these observations, which are certainly shrewd, apt and penetrating, therapist has discontinued the practice of calling on patients in their order around the table, and of requesting a recapitulation of what they have read. Instead, he calls on patients in no particular order. Usually some member of the group will break in with an observation of his own at the conclusion of a passage, or the therapist will amplify and give collateral examples demonstrating the subject.

Frequently patients become so involved in the subject matter that they ask to borrow a book to read at home or on the ward, or inquire where they might purchase a copy and one patient facetiously suggested stealing one. Rarely will a patient protest against the "big words" used in the book, but when he becomes interested in the text his protests subside. In one patient (subsequently undergoing lobotomy) this protest was persistent, but here the reason could be seen in the patient's psychodynamics. A pretty young woman of 22 she felt compelled to derogate herself as ugly, too tall, cross-eyed, coarse and uncouth, all part of the defense of a strongly-fixated oedipal situation.[3] Thus the "big words" were one of her special pet peeves. Most patients, however, make no complaint on that ground.

The fear of polysyllabic diction has assumed the proportions of a widely current stereotype, expressed in an almost universal cliche, and the greatest tenderness for patients seems to be expended to protect them from the "big words" and not to go "above their heads." It is highly questionable whether the danger and damage to patients of exposing them to data "above their heads" is any greater than submitting them to material "below their heads." It is to be suspected that even very regressed patients can be set into vibration with spasms of autistic laughter by the very inanity of insincere, condescending and oversimplified mental pabulum.

It is quite possible another way could be found to interest even the obstreperous lobotomees, but in the long run it seems pointless to struggle and writhe in order to invent some method whose great advantage would be that it avoids the use of a text.

The suspicion and aversion toward the polysyllabic diction derives from two principal sources. First, the objector's own insecurity with regard to the diction and his own state of language facility. Secondly, it stems from our commercial society where one has to "sell himself" and must not be thought to "high-brow" a "prospect." Commercially, it is one's duty to stay on the level of the customer, but therapeutically, the ob-

ligation would seem to be to raise the patient to the therapist's level. The bias against the "big words" seems to lead logically to the belief that anything, no matter how complex and recondite the material, can always be reduced to first grade primer simplicity and monosyllabic diction. Of course, whatever is written and spoken had preferably be as simple as possible—but not at the expense of meaning. Attempts of this kind for patients which the author has perused invariably reduce themselves to the utterly platitudinous, and hence almost useless. Is it not to be assumed with human beings that the exploration of all possible facets and phases of a problem or question will help motivate behavior and illuminate alternative courses of conduct, so that a good choice can be made? At the platitudinous level of exposition such illumination is not possible. The lack of such conceptual foundations for behavior is all too apparent clinically in the disorganized lives and personalities of patients and others.

It can only be concluded here that difficulties of understanding the text reside not alone (and even not at all) in the words used, but much more in the concepts propounded, and it has been found rather constantly that where the material of the textbook engages the affective capacity of the patient, there the bar of language is a minimal hindrance to understanding. Moreover, therapy does not entirely depend on the amount learned. Patients who make progress will be found to have absorbed, by a kind of affective osmosis, more of the spirit of the content than they might be credited with at first glance.

At any rate, patients treated by means of textbook-mediated group psychotherapy do not seem nearly as perturbed over the polysyllabic diction as their would-be rescuers and defenders. On the contrary, they often evince surprising degrees of a reawakening of intellectual curiosity, and, to date no better means appears to be available for husbanding and enhancing emotional responsiveness. Even with the most regressed lobotomees one at least notices a greater degree of social responsiveness, and ward behavior, as reported by ward personnel is distinctly improved.

There is still another great fear which requires our best efforts to pacify. How can the process of learning be thought of as so alien to therapy? And if learning and therapy be in any way related what sacred cow has been violated in the recourse to a book as intermediary and vehicle of therapy? Some acknowledgement of the relationship can be found in a comparatively recently coined term, "bibleotherapy." On this point Powell[1] states: "Education and therapy are complementary phases of a single process—the *learning* process as directed to the achievement of effective maturity. The extent of their identity has been obscured by their more salient differences in emphasis: the one upon mastery in the handling of *judgments*, the other in the handling of *feelings*. By an oversimplified aphorism, we could say that education teaches the individual how to examine *what* he thinks about what he feels important; therapy to examine *how he feels* about what he thinks important. . ."

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Complications in the Use of Pitressin in Schizophrenia

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In recent publications[1,2] the preliminary results of treatment of chronic schizophrenics with pitressin were presented. At that time, no physically untoward effects had been observed during the continuous administration of this hormonal preparation. However, a sudden death which occurred in one of the patients who was receiving this hormonal treatment directed our attention to further investigation. In particular, as in this case, the blood urea nitrogen was raised and we wished to find out more exactly the relation between this finding and any other unexpected symptoms that other patients had shown while undergoing this treatment. The purpose of this paper is to present some of the data obtained on these patients so as to acquaint other investigators with possible hazards of this treatment and to arouse the interest of some group better equipped with laboratory facilities for a study of the action of this hormone.

The techniques were those presented in the previous papers.[1,2] Ten units of Pitressin (Parke-Davis) were injected for the first 10 days, following which 10 units were given every second day.

In the death cited, a 60 year old woman, who had been an inmate of state hospitals for 13 years, was started on pitressin. Physical examinations had been negative, although there was some suspicion as to whether the patient might have had a peptic ulcer. No crater, however, could be demonstrated on two roentgenologic examinations. According to reports on urinalysis, in 1938 this patient had 2 plus albumen; in 1940, trace of albumen; in 1941, trace of albumen in her urine; however, with one exception the specific gravity of her urine was above 1,020. There were no records of further urinalysis between 1941 and the present date.

Twenty days after treatment was begun, patient suddenly collapsed and her blood pressure was unobtainable. Her urine was concentrated and scanty. Urinalysis on the day of death showed 4 plus protein, consistent with the prolonged shock, and numerous white blood cells. Thirty-six hours later, patient died, after having remained in shock following the original collapse. Unfortunately an autopsy could not be obtained.

This death could, of course, have been due to one of the accidents to which people of this age are subject, such as a coronary occlusion, or cerebral vascular accident. The suddenness of the collapse made these possibilities more likely than a uremic episode, or a disturbance of homeostasis due to electrolyte or water imbalance, both of which should have given some preliminary signs.

One hundred and fifty-five patients were subsequently followed and were observed for a period of two weeks before beginning treatments, during which time blood pressures were recorded, urine analysis, and blood urea nitrogens were determined. Blood pressures were observed three times daily and monthly checks of the urea nitrogens and urines were made. Because of the limited laboratory facilities at a state hospital, it was not feasible to determine sodiums, potassiums, phenolsulphonphthalein excretions, and other tests which would be desirable in view of the present lack of knowledge concerning the effect of chronic administration of pitressin. The majority of these patients were in the 4th, 5th, or 6th decade of their lives.

Eight patients out of the total of 155 showed an eventual rise in their blood urea nitrogen levels of a value of over 20 mgm. This level was arbitrarily selected as being significantly pathological. Table I summarizes the blood urea nitrogen in the 5 patients whose treatment was terminated.

From North Carolina State Hospital, Butner, N.C.

TABLE I

BLOOD UREA NITROGENS IN MGM. %

Case No.	Before Treatment	Months Following Start of Treatment					
		1.	2.	3.	4.	5.	6. 7.
1.	12.2	—	17.3	10.4	21.2*	12.2	7.2
2.	—	27.3	20.8	19.4	14.5	12	
		36.6*					
3.	20.3, 18, 13.4	24.8*	17.7	—	19.3		
			15.2				
4.	14.6, 12	27.3*	15.6	—	12.4		
5	18.8	—	9.6	—	9.8	27.2*	14.2 12.4

*Treatment Discontinued.

Of the 5 patients, only No. 3 revealed any abnormalities on physical examination, which in her case were those compatible with hypertensive cardiovascular disease, all five showed a trace of protein in their urine at some time, two never showing more than a trace, and three showing larger amounts, but not consistently. By comparison, of the remainder of the patients given pitressin who fall in the same general age range, only 65% showed at least a trace of albumin at some time or other as compared with 100% of the patients developing elevated nitrogen levels. Whether this is a definite indication of renal pathology is open to question since the urines obtained were all voided specimens and thus subject to sufficient contamination to account for certainly traces of protein. It should also be noted that all 155 patients secreted a urine of specific gravity of 1,022 or higher at some time. This specific gravity can be taken as evidence of at least adequate renal function for ordinary metabolism.

Up to the present time, the youngest patient to develop this elevation is 48 years of age. However, most of the earlier patients given pitressin were aged schizophrenics, as mentioned in the previous reports, and it remains to be seen whether younger patients may not get into difficulty as our series of younger patients grows. It would seem to be the aged patients, with possibly some mild pre-existing renal pathology, or other disordered metabolic reserve mechanism, who are likely to show poor tolerance for this form of therapy. However, it is obvious that both the laboratory studies, and the number in the series presented here, are too sparse to definitely support this view.

It should be noted, in all patients where we have had time for a follow up determination the urea nitrogen levels fell after administration of pitressin was terminated.

On a trial and error basis, an attempt was made on some of the patients with elevated urea nitrogens to increase their hydration and see if this would decrease the azotemia. Hydration was not actually forced, but was strongly encouraged, and the administration of pitressin was continued. The results are summarized in table II.

TABLE II

BLOOD UREA NITROGEN IN MGM. %

Case No.	Before Treatment	Treatment period in months					
		1.	2.	3.	4.	5.	6.
2.	—	20.8†	19.4	4.5	12		
6.	21.5	12	6.8	19†	12	16.2	Discont.
7.	12.5	17.4	11.5	20†	19.7	20.0	19.6
8.	15.5	—	—	21.4†	15	21	10.4

†Period of hydration begun.

It would seem from the results of Table II that there is a tendency for the blood urea nitrogen level to fall if the state of hydration is increased. However, the criticism may be applied that the fluctuations of the urea nitrogen level in the entire series of 155 patients were quite wide, and the data of Table II is insufficient to indicate a continuing downward trend. Case No. 7 may have some significance. The urea nitrogen level did not respond to hydration. In our opinion, this may have been due to the fact that this patient had chronic diarrhea. However, it is significant in the light of attempts to theorize on the mechanism involved that increasing the hydration of the patients did not make them worse.

Discussion

While it is obvious that insufficient data is at hand to elucidate the mechanism of nitrogen retention observed, several possibilities may be suggested.

The observation has been made that administration of the antidiuretic hormone is capable of causing fatal water intoxication. Pitressin causes retention of water, and re-

verses the usual synchronous movement of salt and water, in that it also causes increased excretion of sodium and chloride ions. Recent usage of extremely low salt intake diets, such as the rice diet, and especially when combined with mercurial diuretics, has called attention to the so-called "Low Salt Syndrome," which occasionally develops in a patient undergoing this deprivation of sodium chloride. This is characterized by weakness, lassitude, vomiting, muscle cramps, shock, coma, and finally death. Additional findings include a depression of urinary volume, progressive gain in body weight, an elevation of the non-protein nitrogen, fall in serum chloride and sodium. This is more likely to occur in those patients with pre-existing renal disease. It may be, then, that the raised urea nitrogen levels we have encountered may be due to salt depletion and water retention induced by the administration of posterior lobe hormone and that we are actually seeing the early stages of the Low Salt Syndrome. Since our patients are on ad libitum diets, they are not in the same trap as those who are on salt restricted intakes, and thus may partially compensate for the effect of the antidiuretic hormone by varying their fluid and salt intakes.

On the other hand, from a different aspect, any stress, as stated by Selye,[7] is capable of increasing the output of adrenal cortical hormone and this in turn increases nitrogen catabolism.[6] There is a growing mass of evidence in a neglected problem, that there is a close connection between the posterior lobe hormone, the anterior lobe, and thus of the adrenal cortex.[3] Desoxycorticosterone acetate has been found to antagonize the action of pitressin in that it depresses water excretion and promotes salt retention. Thus, some internal mutual dependency of the adrenal cortex might be expected on a teleological basis. Antidiuretic hormone is reported to cause sodium retention in cats with adrenal cortical insufficiency. Roemmelt et al.,[4] points out that the posterior pituitary hormone is uninhibited in the absence of the adrenal cortex, leading to loss of sodium. Gaunt[3] quotes Lloyd in some unpublished data as finding a drastic fall of serum sodium from chronic administration of pitressin in humans.

One of Gaunt's students has approached the problem from another viewpoint, to determine whether administration of antidiuretic hormone might not be capable of increasing the output of cortical hormone. While her results were inconclusive, in view of the unphysiological doses required to elicit this response, they were at least suggestive that such a relationship might exist. The idea is advanced that this relationship may function through response of both hormonal systems to the serum sodium level.[3,5,8,9] Such an increase in cortical hormone would of course be expected to cause an increase in nitrogen catabolism as demonstrated by many workers.

Antidiuretic hormone is inactivated mainly by the liver,[3] and cirrhotic patients are not capable of deactivating this hormone. If we recall the association of liver disease with schizophrenia, the possibility that this may play a part in the foregoing cases can be seen.

From a somewhat opposite standpoint, the results summarized in Table II would tend to indicate that water intoxication or the "low Salt Syndrome" were not the mechanisms involved. Increasing the hydration of the patient could have been expected to precipitate a crisis, if the patient were in a state of salt depletion. Pitressin, by its vasopressor activity, might be causing a renal shutdown. Thus, hydration might be capable of diluting the pitressin concentration in serum and in this way allow the filtration rate to increase and therefore permit increased excretion of nitrogenous products.

Summary

1. A death which might possibly have been due to pitressin administration stimulated these investigations.

2. Eight patients out of a total series of 155 showed elevations of their blood urea nitrogen levels over 20 mgm.% during pitressin administration.

3. Withdrawal of pitressin was followed by a fall of the urea nitrogen level.

4. Some of the possible mechanisms of this nitrogen retention are presented.

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Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

CASE NO. C-5252

Presentation

A 7-year-old colored boy entered the hospital June 11, 1952, with agranulocytosis, his seventh hospital admission. He died 6 days later.

Birth and development had been normal. He was first brought to the hospital clinic at the age of 5 years on August 14, 1950, because of anorexia and intermittent attacks of abdominal pain which had been occurring over a period of 2 weeks. He had slept more than usual for 1 week. For 3 days he had complained of headache and the parents had observed that his gait was strange. For 2 days there had been right facial weakness and a slight nasal quality to the voice. There had been no difficulty swallowing.

Examination of chest and abdomen was normal; the temperature was 99° F. There was right facial weakness. Ocular movements were normal; the right pupil was slightly larger than the left. The neck was supple. The right arm and leg were slightly weaker than the left. Abdominal and cremasteric reflexes were weaker on the right than on the left. Deep reflexes were equal. There was a questionable Babinski sign, bilaterally. Kernig, Brudzinski and Rhomberg signs were absent. Finger-to-nose and heel-to-shin tests were normal.

Because of bed shortage he was sent home to return to the clinic the next day. Lumbar puncture was performed but the pressure could not be evaluated because of struggling. The fluid was clear and 25 mononuclear cells per cmm. were found; protein was 15 mg. per cent; chlorides 124 meq. per litcr; Wassermann anticomplementary. He was returned home and the parents were instructed to observe him closely. A diagnosis of acute poliomyelitis was reported to the Health Department.

Weaknes of the right arm increased and he was admitted to the hospital on August 21, 1950. The tongue now deviated to the right. The neck remained supple. The right arm was flaccid and reflexes were absent. The right leg remained weak. No weakness was found in the left arm or leg. A Brudzinski sign was obtained and extensor plantar responses elicited on the right. He remained alert. Rectal temperature did not exceed 100.5.

Weakness of the right leg had increased on the second hospital dey and he exhibited a peculiar stare, although ocular movemeints were not impaired. The Babinski sign was present bilaterally. Breathing and swallowing were normal. Muscular tenderness of the right arm and leg was elicited. The ocular fundi were normal. In the afternoon of the same day a neurologist found the pupils to be dilated. Evaluation of vision was difficult but there was impairment, and searching nystagmus was observed. On command the eyes were moved easily to the right but not to the left. Downward gaze was efficient but upward gaze was not. Weakness of the right limbs without increased tonus remained. No sensory abnormality or muscle tenderness was found. Plantar reflexes were extensor. The patient remained alert and cooperative.

On the following day he was completely blind, unable to distinguish light and dark. The pupils were dilated and did not react to light. There was little change in other signs. An ophthalmologist confirmed the complete blindness; he found paradoxical response of the pupils to light and slow hippus of the left pupil. The fundi were normal.

Cerebrospinal fluid a week after entry showed 12 mononuclear cells per cmm.; protein 15 mg. per cent; chlorides 128 meq. per liter; Wassermann negative. A collodal gold test was not performed.

Blood counts were normal and sickling preparations negative. Urinalyses were normal. X-rays of skull, long bones and pelvis were normal.

He remained blind. By September 8 the strength in the right arm had greatly improved and weakness of face, tongue, and right leg had almost disappeared. He was dicharged the following day. At home he ate well and had no complaints for 4 days, but then vomiting recurred, he became unsteady on his feet and was lethargic. These symptoms continued and the parents brought him to the hospital September 15. The rectal temperature was 98.6. His gait was so unsteady that he had to be supported when walking. He constantly fell toward the left.

The neck was supple. He remained blind. Pupils reacted to light but exhibited hippus. Right facial weakness persisted and the tongue deviated to the right. No sensory changes were elicited. The right arm and leg showed slight weakness. He was happy and alert, almost euphoric. Speech was thick and slow.

An electroencelphalogram was considered borderline but probably within normal limits. No seizure discharge nor definite localizing phenomena were observed. Lumbar puncture produced clear fluid with an initial pressure of 140 mm. of water, free of cells; protein 8 mg. per cent; sugar 73 mg. per cent; chlorides 129 meq. per liter; Wassermann and gold curve were negative.

The patient apparently remained blind and with no evidence of localization or increased intracranial pressure he was discharged after a week in the hospital. He improved after discharge. He was able to walk about the house and recognized common objects held in front of him. However, on October 7 he suddenly slumped to the floor and was unable to stand.

He returned to the hospital October 14. He was alert and cooperative. Examination showed severe ataxia. He was unable to sit or stand or to hold up his head. The tongue deviated to the left. The pupils were small and equal, reacted to light and on convergence showed hippus. There was no nystagmus, he was able to follow a moving light, and the examiner concluded that partial vision was present. Corneal reflexes were normal. The eyes wandered without purpose. By October 30 he showed improvement of strength and vision, but ataxia was marked. He was discharged November 4.

He was seen in the clinic where it was noted that ataxia was becoming less. Bilateral Babinski signs and unsustained clonus were elicited. Coarse horizontal nystagmus was observed. An electroencephalogram showed diffuse random slow dysrhythmia.

He returned to the hospital December 6 because of vomiting, anorexia and vertigo of 2 days' duration. He was lethargic, spoke haltingly, complained of headache, and held his head turned to the right. Vision was poor with central scotoma and there was nystagmus on left lateral gaze. The ocular fundi were normal. Motor function of the extremities appeared normal. Bilaterally hyperactive deep reflexes were elicited. Babinski, Chaddock, Oppenheim signs and sustained ankle clonus were found bilaterally.

He improved with hydration. Ankle clonus disappeared briefly only to reappear on the following day. Incoordination of the left arm appeared on December 11. Five days later left hemiparesis appeared and ankle clonus was inconstant. He required support to stand in bed. Heart and lungs were normal.

On December 11 lumbar puncture with fair relaxation showed an initial pressure of 170 and final pressure of 110 mm. of water after removal of 8 cc. The fluid was clear and colorless with 10 cells per cmm., mostly lymphocytes; protein 14 mg. per cent; sugar 58 mg. per cent; chlorides 125 meq. per liter; Wassermann, gold curve and culture were negative.

Severe ataxia of the left arm and leg were present on December 14. He was able to recognize his doctors. Pupils reacted normally to light. He was discharged December 16 and followed in the clinic, where he showed general improvement.

He returned to the hospital on February 3, 1951, with night crying because of headaches and a behavioral change with much obscenity. For about a week he had had to strain to urinate. In the hospital he was cooperative but petulant. He had lost his friendly alertness and he perseverated. The head was rigidly rotated toward the left. Pupils were 6½ mm. in diameter and reacted promptly to light and on convergence. Vision was severely impaired but he could distinguish colors. His speech was a monotone. Plantar responses were strongly extensor to almost any stimulus. Deep reflexes and abdominal skin reflexes were hyperactive. Cremasteric reflexes were absent. A neurologist found him in worse mental and physical condition than on previous examinations. He attributed the nuchal stiffness, which was present in the reclining, sitting and standing positions, to imbalance of head turning centers.

An electroencephalogram February 13 showed non-focal slow dysrhythmia, more disordered than the record of November 1950.

Cortisone was administered from February 15 to March 6. During this period edema of ankles, legs and face appeared. Following the cortisone he became more alert and cheerful and vision improved. He left the hospital in March and was followed in clinic. Mentality improved and he was again able to walk although ataxically. In June there were left-sided convulsions without aura or cry, lasting for a minute, occurring several times a day. Frequency of seizures decreased with the use of ½ grain of phenobarbital every 8 hours.

There was general improvement for several months; he walked with more assurance, fell infrequently, showed improvement in speech, but mental retardation and general overactivity remained. Left sided weakness continued.

The seizures recurred in March 1952 and the dosage of phenobarbital was increased without effect. In addition to the seizures he began to have episodes in which there was lateral flexion of the head and trunk to the right with extension of the left arm, accompanied by a "dreamy look." The duration was only a few seconds and he did not fall. Tridione therapy was instituted May 7, 1952, with dosage of 0.3 gm. 3 times a day.

Electroencephalograms were interpreted as follows: The record of March 12, 1951, showed no change over that of a month before. June 29, 1951: Random slow waves and much fast activity in all leads. Isolated high voltage spike waves from the right occipital area. August 7, 1951: Slow disorganization. Fast activity modulating background, thought to be barbiturate effect. April 3, 1952: Record under sedation—random slow dysrhythmia, non-focal, modulated by barbiturate effect.

He developed fever, anorexia, vomiting and a stiff neck on June 9, 1952. Tonsils and pharynx were quite red. The chest was clear. Rectal temperature was 104.4. A cerebrospinal fluid cell count revealed 2 mononuclear cells per cmm. He was given penicillin and gantrisin. He returned to clinic the following day for an injection of penicillin. Rectal temperature was 105.4. In the clinic on June 11 the rectal temperature was 104.2. The pharyngeal inflammation was no better. He was lethargic and dehydrated. A white blood cell count was 3,200 per cmm. and no granulocytes were found. He was admitted to the hospital and given small transfusions, chloromycetin and penicillin. The white cell count fell to 1100 per cmm. Blood non-protein nitrogen rose to 57 mg. per cent. He gradually weakened and died on June 17, 1952.

Differential Diagnosis

Dr. Charles D. Aring: I would first like to dismiss poliomyelitis as a consideration in this case. I feel certain that the students did not make this diagnosis after they had read the protocol, but since it was considered early in this boy's illness, I believe it well to say that as it developed this could not have been the picture of poliomyelitis.

Our patient had a disorder producing mental, cranial nerve, motor and reflex abnormality without too much in sensory spheres besides the blindness and of course some muscle tenderness from time to time. I would like to note for reference later that the signs of central nervous disorder arose from lesions above and below the tentorium. The tentorium, you will recall, is that reflexion of the dura that separates the middle from the posterior cranial fossa. In other words he had signs of disease in the cerebral hemispheres as well as signs arising from the cerebellum and brain stem. This may help us in differential diagnosis.

We visualize from the protocol a steadily progressing and remittent disorder of the central nervous system whose identifying features are discreteness and multiplicity of lesions and consequent widespread signs and symptoms. There was reversibility of the lesions in the earlier stages, permitting some temporary recovery from the symptoms, but there was a gradual progression of the disease toward disability. So, I think we could define this disorder as a disease of remissions and exacerbations scattered in time and space—central nervous system space of course. Now I believe that none of us would hesitate to make the diagnosis of multiple sclerosis here if it weren't for the age of the patient and indeed this is the diagnosis that I made when I saw this lad in the first of 2 or 3 consultations at his age of 5 years on August 21, 1950. It is not well-known perhaps that it is possible to have multiple sclerosis in infancy and in childhood. Therefore, I've set forth a compilation taken from Wilson's encyclopedic text of neurology, of more than 1100 cases of multiple sclerosis, depicting their ages at the onset of symptoms:

Age in Years	*Per cent*
Below 10	2.2
10-20	14.0
21-30	31.8
31-40	28.0
41-50	13.3
51-60	4.2
Over 60	0.3

You will observe that in about 2 per cent of patients the age of onset was below 10 years. The onset in multiple sclerosis has been reported as early as 3, 4, and 5 years and at the polar extreme there is a well-authenticated case on record of a patient who had the onset of symptoms at the age of 81 years. I myself have seen a patient whose first symptoms began at the age of 70 although he did have temporal optic atrophy which had been symptomless.

So I believe that this boy had multiple sclerosis. Now what might we say about other demyelinating diseases? I have also set forth the names of the commoner demyelinating conditions, that is, the eponymic designation for these conditions; such classification of Schilder's disease as may be found in the textbooks.

Schilder's Disease

Acute infantile type—Krabbe
Subacute juvenile type—van Bogaert-Scholz
Chronic type—Pelizaeus-Merzbacher

We should, of course, consider whether or not this patient had Schilder's disease, if that disease can be considered an entity. Schilder's disease is also termed encephalitis periaxialis diffusa, also diffuse sclerosis, according to the neurological tenet of not using one term where two or three will do. In this condition there occurs a more or less symmetrical destruction of white matter associated with a clinical syndrome of no great uniformity. This destruction peculiarly enough spares the so-called "U" fibers, that bit of the white matter immediately subjacent to the cortex which connects one gyrus with another. However, the deeper white matter—the long fiber systems—is destroyed in diffuse sclerosis or Schilder's disease. Where this destruction begins, whether anteriorly or posteriorly or centrally, depends the symptoms. In my experience this most often begins posteriorly and we see first a rather complete blindness. Whether or not this condition, Schilder's disease, is an entity is still being argued. It may represent a variant of multiple sclerosis, and I'll discuss that point further as we go along. First I would like to take a moment to define the terms that I've been using.

Encephalitis periaxialis diffusa obviously means a disorder of the cerebrum which is diffuse in which the axones are demyelinated, periaxialis referring to that portion of the axone surrounding the fiber itself, in other

words demyelination. This term is not correct since in Schilder's disease as indeed in multiple sclerosis we do see destruction of axones. True, the myelin bears the brunt of the lesion but there is axonal destruction too. The term diffuse sclerosis arose because in certain instances the astroglia hypertrophy. But this is not always the case in Schilder's disease, and therefore this term too is not quite correct from a semantic point of view. Perhaps a diffuse and progressive leukoencephalopathy would be the better designation.

Now to turn to the problem of whether or not Schilder's disease is an entity—whether this demyelinating encephalopathy can be separated from multiple sclerosis—I'd like to present some evidence that perhaps it might. In the first place the size and distribution of the lesions in Schilder's disease and their sometimes cystic structure, is thought by many neurologists and neuropathologists to justify a separate description for Schilder's disease. Other points of possible differentiation are as follows:

2. Age. Schilder's disease notoriously occurs in infants and children. The age of 15 is a usually accepted dividing line; some 50 per cent of the cases have their onset before the age of 15, and some 50 per cent are older. So there is this frequent childhood development, in contra-distinction to what we see in multiple sclerosis.

3. The frequent familial incidence is well-known in Schilder's disease. While multiple sclerosis has been reported in families, it's rather unusual to see more than one member of the family afflicted.

4. The signs in Schilder's disease arise from lesions located above the tentorium. In multiple sclerosis sub-tentorial signs occur frequently but in Schilder's disease the massive destruction of white matter is located in the cerebral hemispheres where the clinical manifestations are found.

5. Blindness may begin soon and become complete in Schilder's disease. In multiple sclerosis complete blindness is practically never met with. By complete blindness I mean persistent and complete. As you learned our patient seemed completely blind at one stage, this happens with fair frequency in one eye in patients with multiple sclerosis, but I can recall the report of only two cases of the thousands with multiple sclerosis who had suffered complete blindness.

6. Mental manifestations are more evident earlier, are more severe and can lead to frank dementia in Schilder's disease.

7. Sensory symptoms are commoner in multiple sclerosis than in Schilder's disease.

8. Schilder's disease is more commonly marked by generalized convulsions than is multiple sclerosis. So if we review these points of differentiation, trying to make a diagnosis in our own patient, I think we come reasonably to the conclusion that if Schilder's disease is an entity, then our patient has multiple sclerosis.

Since convulsions were indirectly responsible for our patient's death, I would like to discuss this aspect of the problem to the exclusion of the more usual symptoms and signs.

This boy had, as you recall, a series of Jacksonian fits which may occur early or late in multiple sclerosis. They usually herald a paralytic condition or they involve musculature already weakened. In our patient we see that the weakness was at its greatest in the left limbs beginning on or about December 16, 1951, and that these left-sided convulsions were occurring in June of 1952. During the left-sided attacks the boy was suffering a left hemiparesis.

Generalized fits are by comparison distinctly rare in multiple sclerosis and petit mal rarest of all. In symptomatic epilepsy—convulsive seizures from a known cause—it is not unusual to see partial fits, that is, instead of the complete fit occurring we see bits of it. This is what I surmise was happening with this boy. What I'm getting to is that I don't get the feeling that this lad was suffering from petit mal, and this brings me to the agranulocytosis.

In a general hospital or a consultation practice of neurology I myself have never used drugs that I know will produce blood dyscrasias. I did not say that the alert practitioner should not use them. (As someone has aptly put it: "Don't quote me, that's not what I said, that's what you heard.") Of course they should, but in a general hospital practice of neurology, patients sort of fall

between the interstices. In a large teaching hospital where it is necessary to compromise between patient care and teaching, the patient usually does not see the same physician more than several times running. The use of these potent substances lays upon you a great responsibility, much greater than the prescription of phenobarbital for example, which while potent, in my experience has never produced a blood dyscrasia. In treating epileptics the oft-advised, frequent white count, is of little or no aid because it would require rather constant titration of the blood in this regard which just isn't feasible. This is another instance of trying to push off on the laboratory the responsibility that is yours. To illustrate how early this tendency is ingrained, my invariable experience with junior student groups presented their first, second, or third neurological case, almost before I have given the history and a survey neurological examination is done, is their inquiry about the Wassermann reaction.

In agranulocytosis, at least in epileptics receiving anticonvulsant drugs, the white count drops precipitously, and while blood counts must reveal the true nature of things, they will not—at least in my experience—propitiate the Gods. Also white counts drop for no apparent reason in people who are not taking drugs. When your patients are taking anti-convulsant drugs, and as you know some offend more than others, it happens to be a responsibility that you cannot escape (1) to instruct the patient or his relatives to report symptoms to you quickly and particularly fever and discomfort in the throat, and (2) to keep ever before you the possibility of granulocytopenia, so that when you hear about the symptom you don't treat it lightly.

I could go into many of the signs and symptoms presented by our patient and also into the cerebrospinal fluid findings, but this would be redundant, so I believe I will close with the diagnosis of multiple sclerosis and agranulocytosis.

Dr. A. A. Weech: There are no roentgenograms made in this case so that we can ask at once for the report of the morning student conference.

Dr. Robert A. Helm: Eighteen students voted for multiple sclerosis and 9 students voted for Schilder's disease. All students agreed that death was due to agranulocytosis from tridione.

Dr. Albert B. Sabin: I think there is one point in the history that should be considered and that is that this child had chickenpox at the end of June 1950. That happens to be about 4 to 5 weeks before the onset of the first symptoms. An interval of 5 weeks between one of the childhood diseases, measles, varicella, etc., and the onset of nervous system involvement is rather long, but since we are almost in complete ignorance of what it is in measles, chickenpox and certain other infections which gives rise, among other manifestations in the nervous system, to demyelinating changes, we also cannot be certain that 5 weeks is too long for the effects to become clinically manifest. This brings us to another consideration in the differential diagnosis. In addition to Schilder's disease, disseminated sclerosis and the "hereditary" demyelinating diseases, we should perhaps also think of postinfectious demyelinating disease. I bring it up here for two reasons: (1) I would like to ask Dr. Aring to indicate what clinical manifestations in this patient would make it possible to differentiate between postinfectious encephalomyelitis and disseminated sclerosis, and (2) to bring up for discussion the pathological differentiation between postinfectious encephalomyelitis, disseminated sclerosis, and Schilder's disease.

In both Schilder's disease and disseminated sclerosis, the lesions are presumably limited to the white matter, if one discounts extension to the gray matter by contiguity, while in postinfectious encephalomyelitis the gray matter and white matter are almost equally affected. The distinction between Schilder's disease and disseminated sclerosis would depend, as Dr. Aring already pointed out, on the distribution of the lesions in the white matter—in Schilder's disease we would expect to find rather extensive lesions in the white substance of the hemispheres, but not in that of the cerebellum, pons, medulla or spinal cord, while in disseminated sclerosis the lesions would be scattered throughout the central nervous system.

Dr. Aring: The differential diagnosis between the infectious encephalopathies and the demyelinating conditions is not difficult with the history of 2 years of remissions and exacerbations. Children who survive the

onslaught of exanthematous encephalomyelitis do not have subsequent attacks; the sequelae, if any, are static.

Children with encephalomyelitis associated with exanthema such as measles are more ill than was this lad; my experience has been almost entirely of a meningitic or meningoencephalitic form. In my experience these things are overwhelming, beginning with fever, drowsiness and disturbed consciousness, signs that are practically unknown at the outset in multiple sclerosis. The nervous manifestations develop out of a general systemic reaction into encephalomyelitis. I know that there is a spectrum of diseases and that certain persons may be touched lightly. Another point is the expectation of increased pressure and cells in the cerebrospinal fluid with the infectious types of encephalomyelitis. In multiple sclerosis the white count rarely approaches 100 cells per cmm., in fact a mild pleocytosis is found in some 30 per cent of cerebrospinal fluids, as was the case with our patient. Of course you may find no cells in the fluids in either condition. It may have been fortunate that we didn't have the story of chickenpox, though I have little doubt that we could have coped with it successfully in diagnostic differentiation.

Dr. M. A. Blankenhorn: Can comment be made on optic atrophy in multiple sclerosis?

Dr. Weech: And what about the condition known as neuromyelitis optica?

Dr. Aring: Neuromyelitis optica undoubtedly is a variant of multiple sclerosis. I should point out that this term is incorrect. As far as I know all cases that have come to autopsy have had lesions besides those in the optic nerve and *myelos* (spinal cord); the lesions are just as disseminated as in any other types of multiple sclerosis. Usually this is a fulminating type of multiple sclerosis which may result in death within days or weeks, sometimes months. Some people survive with it. As far as I am concerned it usually is merely the same disease compressed in time.

Optic atrophy is reasonably common in multiple sclerosis. I don't have percentage figures, but if we include temporal pallor, I would guess that about 50 per cent of these patients who reach me have it. There is a physiological variation in coloration between the nasal and temporal margins of the disc. The blood vessels traverse the nasal side and this makes for a pinker half of the disc. Atrophy of the whole disc is not common. I would guess that partial forms of atrophy of the entire disc may be seen in somewhat less than 5 per cent of patients. However, as I said before, it is remarkable that this practically never results in blindness, but I don't think it's any more remarkable than

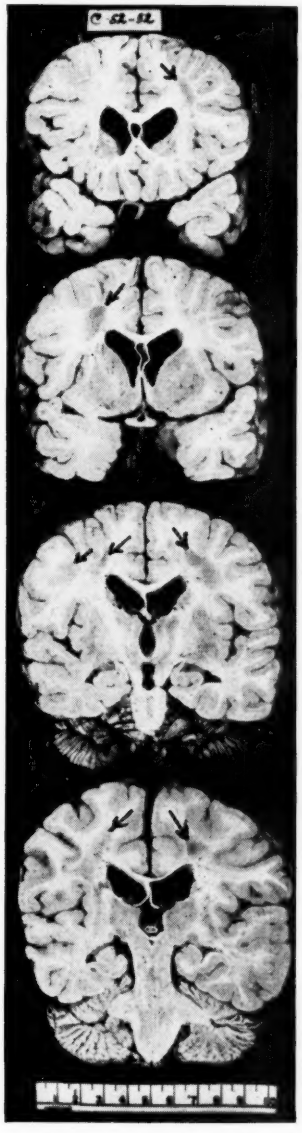

Figure 1. A photograph of several of the vertical sections of the brain to show the slight ventricular dilatation (for a boy aged 7 years), and the larger placques in the white matter (arrows).

the fact that in the dissemination of the lesions of multiple sclerosis, sensory systems are frequently involved yet rarely can we demonstrate objective signs of sensory disturbance in these people, with the exception perhaps of the diminution in the appreciation of vibration. Obviously this means that a demyelinated fiber still has the potentiality of transmission, that it has only been short-circuited so to speak, and that the chance of its transmitting impulses again, are there. Multiple sclerosis is not easily separated from Schilder's disease. Certainly at one stage of this boy's illness his trouble might have been termed Schilder's disease by some neurologists. I am certain he had lesions below the tentorium since he had such an obvious cerebellar syndrome at one stage. Somewhere in the protocol this is well described; the boy had good strength and with it tremendous ataxia. There were other infratentorial signs such as nystagmus.

Discussion of Pathology

Dr. Paul Wozencraft: The autopsy was performed 6 hours after death. The brain weighed 1160 grams. Nothing abnormal was noted on inspection of the external surfaces of the fixed brain and spinal cord. Section of the fixed brain was done one week after the autopsy examination. Inspection of the interior of the brain showed slight dilatation of all the ventricles, which was most evident in the third ventricle. Scattered in the white matter of all lobes were rather large areas of brownish discoloration, firm to touch, and suggesting placque formation. (Figure 1.) Sections of the spinal cord revealed nothing abnormal to the naked eye.

Staining for cells and for myelin sheaths brought out the microscopically numerous lesions scattered throughout the white matter of the central nervous system, sometimes contiguous with blood vessels but more often not. The myelin sheaths bore the brunt of the lesions which in Loyez stains were sharply demarcated from the more normal white matter. Fat-granule cells were numerous in the placques and around contiguous blood vessels; and lymphocytes, plasma cells, microglia and macroglia were involved in the reaction. Placques also were delineated microscopically in stains other than those for myelin sheaths, by the presence of many gemistocytic astrocytes.

These lesions were found in white matter, above and below the tentorium, in the optic nerves, and in the spinal cord where they appeared to be younger and of the so-called "shadow" type, with swollen myelin and ballooning of sheaths.

Next to the agranulocytosis. There was severe ulceration in the cecum. In the cecal mucosa there was abrupt transition from normal tissue into an area of necrosis with edema of the wall. There was no neutrophilic reaction since of course the patient was not able to make neutrophils. In the higher magnification of the area of necrosis in the cecum one sees a few lymphocytes, but no neutrophils whatsoever.

In a section of bone marrow under low power view scattered megakaryocytes and some sickly looking nucleated red blood cells were evident. There were no cells of the granulocytic series to be seen although there were a few scattered plasma cells. A higher power view, showed megakaryocytes in more detail, some scattered nucleated red blood cells and an occasional plasma cell, but no cells of the granulocytic series.

In the course of our study an interesting sidelight was seen—a lesion that Dr. Rich brought to our attention in 1944, found most often in cases of severe overwhelming infection, the so-called tubular degeneration of the adrenal gland. There is necrosis of cells and seepage of fluid into the cords which have the appearance of kidney tubules. This is a degenerative phenomena. Dr. Rich thought that when it was severe and extensive it might account for adrenal failure in some cases of severe infection. The higher power view of the same lesion depicts a remnant of one cell within the lumina of this pathologically produced tubule.

Clinical Diagnosis

Multiple sclerosis.
Agranulocytosis, toxic, due to tridione.

Dr. Aring's Diagnosis

Multiple sclerosis.
Agranulocytosis.

Anatomical Diagnosis

Multiple sclerosis.
Agranulocytosis.

Dr. Blankenhorn: Was there any information to be gathered from studying the bone marrow that would lead to an explanation of the agranulocytosis?

Dr. Edward Gall: You know, of course, that we have no explanation for the development of agranulocytosis; perhaps that's the basis of your question. It has been presumed that the alteration in bone marrow occurring in agranulocytosis is the result of a specific noxious effect on cells which produce granulocytes, whereas as we saw in this case and as we see in so many cases, it is not quite as specific as all that and it effects most of the bone marrow forming elements. Although clinically we are often struck with the disproportionate diminution of white cells as against red cells, nonetheless there is almost invariably a depletion of red cells as well, and in those cases which survive for any length of time red cell depletion may reach the same proportion as white cells but as to what the exact mechanism is I am unable to say, perhaps Dr. Vilter, you can take it from there.

Dr. Richard W. Vilter: I am in agreement, Dr. Gall. The same process may effect the erythroblasts or the megakaryocytes and then later on may effect the white cells. The result in all these situations, if the patient lives long enough, is pancytopenia. There is a lot of speculation about the mechanism, about its being some allergic hypersensitivity process, words which most everybody falls back on when something can't be explained. There has been some evidence, particularly in respect to sulphonamides, that these drugs act as heptanes which in conjunction with protein substances in the body form antigens which stimulate the formation of antibodies. When the drug is readministered an antigen-antibody response takes place, certainly this is no explanation of why bone marrow cells are hit so specifically. I do follow right along with Dr. Gall, the specificity is not against one cell type in the bone marrow but is against all types in varying degrees.

Dr. Blankenhorn: The situation is about what it was 5 years or 10 years ago, except that we've added a few more drugs that may produce agranulocytosis. Suspicion has been cast upon aureomycin and certainly chloromycetin.

Mr. Richard Selzer (Student): I'd like to know if, with any frequency, cases coming to autopsy thought to have acute poliomyelitis, are actually examples of multiple sclerosis.

Dr. Sabin: Those patients dying of acute poliomyelitis usually die within a few days after onset, except as they die as a result of complications in the respirator. It is true that every epidemic of poliomyelitis gathers within the wards devoted to these patients, occasional examples of all sorts of acute neurological disorders, which are not usually winnowed out until the epidemic is over. Now what brought forward the possibility of poliomyelitis in this patient and what was against it? There was no history of fever, but it could have been missed; maybe the parents didn't take the temperature. This boy had pyramidal tract signs early, which is against the diagnosis of poliomyelitis but not entirely so. Babinski signs may be encountered in poliomyelitis but if present they remain for only a day or two and then disappear.

I will take your question as a point of departure to clear up a few points. I found at least 10 reports in the literature of blindness associated with poliomyelitis. When these papers are carefully studied one finds that the diagnosis of poliomyelitis is quite shaky, so that what has been published as fact, has never been established. Because I knew of these reports, we obtained stools from the patient during the acute phase of his illness and innoculated two cynomolgus monkeys. They developed no paralysis, and histiologically there was no evidence of poliomyelitis.

Dr. James V. Glaser: I should like to ask the effect of ACTH on multiple sclerosis. Is it indicated, and does its administration result in a more severe recurrence of the symptoms following the administration of ACTH?

Dr. Aring: We've had some experience in this field and there is enough in the literature to allow us to nudge this question around a bit. It appeared that this lad experienced improvement in his vision after the administration of cortisone. We have seen improvement in patients with multiple sclerosis who have suffered sudden transverse myelitis. In one or two instances we seem to have obtained a rather striking improvement with cortisone, and there are similar reports in the literature. That it makes any difference in the eventual outcome, I would have grave doubts. These substances might be useful in tiding a patient over an extraordinarily difficult and severe stage of the disease. I have no opinion as to whether or not these drugs make for more exacerbation, either more in number or more severe; I don't think we've followed such patients long enough to be able to have an opinion.

Student: Aren't the lesions of the adrenals seen in this boy similar to those described with septicemia as in the Waterhouse-Friederichsen syndrome?

Dr. Wozencraft: I have not been able to find much about them; in Doctor Rich's original report he described one case that was thought to have had the Waterhouse-Friederichsen syndrome. There was no hemorrhage of the adrenal glands but an extensive tubule degeneration but not as extensive as that seen in this case.

Student: Is there any reason to believe that the adrenal cortex extract that is recommended in septicemia, has any effect in preventing the Waterhouse-Friederichsen syndrome from occurring?

Dr. Gall: In the examination of the organs from patients who were treated specifically for meningococcemia with antibiotics alone and from those who had a combination of antibiotic substances and adrenal cortical extract there has been relatively little difference in the manner of their reaction to

my untutored eyes. This tubulization of the adrenal gland may be considered according to the formulations of Selye as a manifestation of a phase in the adaptation syndrome.

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